In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 18-1538V

(to be published)

DURENDA WHITEHEAD *and* KEYNARD SHAWTELL JOHNSON, SR., *on behalf of KSJ, JR.*,	*	Filed: September 29, 2021
Petitioners,	*	Chief Special Master Corcoran
v.	*	
SECRETARY OF HEALTH AND HUM. SERVICES,	*	
Respondent.	*	

R. Christopher Irwin, III, Cook & Tolley LLP, Athens, GA, for Petitioners.

Dhairya Divyakant Jani, U.S. Dep't of Justice, Washington, DC, for Respondent.

ENTITLEMENT RULING[1]

On October 4, 2018, Durenda Whitehead and Keynard Shawtell Johnson, Sr., filed a petition seeking compensation under the National Vaccine Injury Compensation Program ("Vaccine Program") on behalf of their minor son, K.S.J., Jr. (hereinafter referred to as "K.J.").[2] Petitioners alleged that the MMR (measles, mumps, rubella) vaccine K.J. received on January 17, 2017, caused him to suffer from encephalitis and encephalopathy under the Vaccine Table. Petition at 1 (ECF No. 1). In the alternative, Petitioners alleged that K.J.'s receipt of several vaccines on

[1] This Decision will be posted on the Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012). **This means that the Decision will be available to anyone with access to the internet.** As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the Decision's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public in its current form. *Id*.

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter "Vaccine Act" or "the Act"]. Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

January 17, 2017 (including the MMR, influenza ("flu"), hepatitis A, diphtheria, tetanus, acellular pertussis ("DTaP"), hepatitis B, inactivated polio vaccine ("IPV"), haemophilus influenza type B ("HiB"), pneumococcal conjugate vaccine ("PCV12"), and varicella vaccines), caused him to suffer "the activation of his SLC19A3 gene variant," leading him to experience an "SLC19A3-related encephalopathy." Petitioners' Prehearing Brief, filed Nov. 19, 2020 (ECF No. 32) ("Prehearing Brief").

An entitlement hearing was held in this matter on March 4, 2021. Having reviewed the materials filed in this case and considered the parties' arguments, I hereby find that Petitioners have met their burden of proof, and are therefore entitled to damages. Although Petitioners have not met the evidentiary requirements for a Table encephalopathy injury, they have established preponderantly that the multiple vaccines K.J. received in January 2017 likely triggered his preexisting genetic mutation, manifesting as a specific disorder: biotin-thiamine-responsive basal ganglia disease ("BTRBGD").

I. Medical History

Pre-Vaccination History

K.J. was born on November 30, 2015. Ex. 3 at 13. At his birth, K.J. weighed 6 pounds, 1.3 ounces, and his Apgar score was 9 at one minute and 9 at five minutes, although he failed his newborn hearing screening. *Id.* at 12; Ex. 5 at 33. During his first two months of life, K.J. had regular well-child checks with his pediatrician at four days, two weeks, one month, and two months of age, respectively. *See* Ex. 2 at 15–22. During that time, he was found to be healthy, and noted to be growing and developing normally. *Id.* On February 5, 2016, at his two-month-old well-child check, K.J. was administered certain routine childhood vaccines without incident. *Id.* at 16.

Prior to the receipt of the vaccinations at issue in this case, K.J. was hospitalized three times for respiratory-related conditions. *See* Ex. 7 at 167; Ex. 5 at 83–87; Ex. 6 at 10–13. On April 19, 2016, K.J. was hospitalized overnight for bronchiolitis, but discharged the next day. Ex. 7 at 167. A few months later, on June 9, 2016, K.J. presented to the Coliseum Medical Center Emergency Department in Macon, Georgia with another case of bronchiolitis. *See* Ex. 5 at 83–87. He was treated with bronchodilators and transferred to Scottish Rite Hospital in Atlanta, Georgia, where he remained until June 14, 2016. *Id.* at 87–88; Ex. 7 at 93. As part of his evaluation during this hospitalization, K.J. had an abnormal swallow study. Ex. 7 at 467. His diagnoses were bronchiolitis, respiratory failure, and laryngomalacia. *Id.* at 93. Finally, on November 18, 2016, K.J. was again hospitalized overnight for respiratory distress and a history of laryngomalacia. Ex. 6 at 10–13. He responded well to steroids and bronchodilators, and was discharged the following day. *Id.*

Vaccinations and Onset of Alleged Injury

On January 17, 2017, K.J. saw his pediatrician for a one-year-old well-child check. Ex. 2 at 12. No abnormal findings were noted on physical examination, and he was administered several routine childhood vaccinations, including the MMR, flu, Hepatitis A, DTaP/HepB/IPV, Hib, PCV13, and varicella vaccines. *Id.* at 13.

Five days later, on January 23, 2017, K.J. was taken to the Athens Regional Medical Center Emergency Department for concerns of generalized weakness, fussiness, poor oral intake, and wet diapers. Ex. 8 at 13. At that time, Ms. Whitehead provided the following history:

> [He] was behind on his vaccine/immunizations and one week ago [he] had multiple vaccines to help catch him up on his immunization. Mom reports the child did well throughout the week but over the weekend mom noted that the child has had difficulty with crawling and ambulating. He just started cruising/walking over the last few months and he has been meeting his milestones. Mom reports he appears uncomfortable and seems to have pain with attempting to crawl and ambulate.
>
> Mom has also noted that he seems to be weak in the arms. She reports that when he reaches out for things he will drop his arm. [S]he states she [ha]s also noted that [at] times he has a fine tremor in his arms when he is trying to reach out for objects. No fever. No seizure activity. No nausea or vomiting.

Id. Despite such concerns, K.J. was awake and alert with good eye contact, and had no fever, rash, or respiratory symptoms. *Id.* at 13–14. In addition, his vitals were stable, and he did not appear to have any acute infectious process. *Id.* at 14.

As part of his initial evaluation, K.J. underwent a brain computed tomography ("CT") scan, which demonstrated "[m]oderate low-attenuation in the basal ganglia bilaterally, having a symmetrical appearance, plus areas of low-attenuation in the cerebral white matter, having a parasagittal distribution on the right." Ex. 8 at 15. The scan also revealed several lesions. *Id.* at 14 It was noted that if these findings were "of acute onset," the differential diagnosis would properly include "hypoxia, encephalitis, and carbon dioxide exposure," whereas if the findings were "more chronic," the differential diagnosis should include "demyelinating disease and a number of congenital disease processes." *Id.* at 15–16.

K.J. was subsequently transferred to the Scottish Rite Hospital that same day in January 2017 for a higher level of care and further work-up and treatment. Ex. 8 at 14. There, Ms. Whitehead again provided a medical history overview. In it, she recounted that K.J. had "woke up and seemed normal" two days before (which would have been January 21, 2017), but then would

not rise when called upon, and had trouble reaching toys placed in front of him. Ex. 7 at 3107. He also proved unable to hold his bottle and seemed weak and lethargic. *Id.* By the next day (January 22, 2017), he seemed especially "spaced out" and sleepy, and so Ms. Whitehead allowed him to sleep through the night before taking K.J. to the Emergency Department the next day. She added that K.J. had not experienced any recent fever or upper respiratory infections. *Id.*

Upon examination, K.J. was active and alert, and displayed normal reflexes or cranial nerve deficits, although he was unable to sit or stand without assistance and was mostly limp. Ex. 7 at 3107. The examining physician commented that there were no obvious differences between his right and left side, and it was unclear whether K.J.'s condition represented ataxia or weakness. *Id.* On January 24, 2017, K.J. underwent brain and spine magnetic resonance imaging ("MRI"), and the brain MRI revealed "symmetric areas of abnormal signal, diffusion restriction, and abnormal enhancement" that involved "the basal ganglia, thalami, and cerebral cortex." *Id.* K.J.'s treating physicians noted that these findings were "concerning for metabolic vs. postvaccine encephalitis vs. ADEM [acute disseminated encephalomyelitis]." *Id.* Based on this initial exam and testing, treating physicians recommended that K.J. be transferred to the pediatric intensive care unit ("PICU"), which Petitioners initially refused but later accepted, and K.J. remained in the PICU until February 6, 2017, when he was transferred to inpatient rehabilitation. *Id.* at 3108, 3110.

Specialist Evaluations in 2017

During his PICU admission, K.J. underwent several tests and medical evaluations. Electroencephalogram ("EEG") testing reflected normal results in the awake, drowsy, and sleep stages, with no epileptiform abnormalities or electrographic seizures. Ex. 7 at 3932. On January 25, 2017, K.J. underwent an infectious disease consult with Lisa Cranmer, M.D., who noted that it was unlikely that K.J. suffered from viral or bacterial encephalitis given the absence of fever, pleocytosis (increased white blood cell count), and other viral symptoms. *Id.* at 3197. Dr. Cranmer also listed possible non-infectious differential diagnoses, to include metabolic complications. *Id.*

That same day K.J. had a neurology consult with Rebecca Luke, M.D., who discussed the possibility that K.J. was suffering from ADEM, but discounted its likelihood, since his brain MRI did not reveal the presence of white matter involvement typical of ADEM. Ex. 7 at 3276. Laboratory testing performed at that time revealed an elevated serum lactate and pyruvate levels, with normal ammonia levels. *Id.* at 24. K.J. had a follow-up visit with Dr. Luke on January 29, 2017, and she now observed that K.J. was tracking people in the room and seemed less irritable. Ex. 7 at 3446. K.J. was placed on a regimen of thiamine and levocarnitine for treatment of a possible mitochondrial disorder. *Id.*

By the morning of February 6, 2017, K.J. "was showing some mild improvement—he was reaching for toys, and seemed more alert although he remained intermittently irritable." Ex. 7 at

3446. He participated in inpatient rehabilitation, including physical therapy, occupational therapy, and speech language pathology until his discharge on February 21, 2017. *Id.* at 3116–17. At discharge, the "predominant differential [diagnosis] was mitochondrial disease triggered by administration of vaccines." Ex. 11 at 34. Laboratory specimens were sent to Baylor for mitochondrial genome sequencing. *Id.*

The following month, K.J. was seen by a speech language pathologist, Jennifer Ponder, on March 6, 2017. Ex. 7 at 56. Ms. Ponder recorded that the family had no concerns about K.J.'s eating at that time, although they were still taking some precautions in feeding. *Id.* Ms. Ponder did not observe any overt signs or symptoms of laryngeal penetration or aspiration. *Id.* By March 17, 2017, K.J. had started walking with the assistance of a walker. *Id.* It is not evident from the records at this time whether K.J. was still continuing to receive thiamine.

On April 18, 2017, K.J. was admitted to Navicent Health Medical Center for evaluation of worsening ataxia and irritability. Ex. 11 at 33. In particular, the Petitioners had observed that a week before, K.J. had "started becoming more clumsy (again…) and [was] falling a lot more. He also started becoming remarkably hypotonic with interval inability to sit on his own or by himself. He also started becoming more irritable" and "having low grade fevers." *Id.* In addition, on the previous night (April 17, 2017) K.J. had "an episode of disorganized perioral twitching that was not associated with impairment of level of consciousness." *Id.* An EEG revealed "diffuse delta activity without subclinical seizures." Ex. 11 at 33.

A neurological exam by Joseph Trasmonte, M.D., showed that K.J. was "[v]ery irritable. Noninteractive. [Exhibiting a] [h]ead lag. Unable to sit on his own…Tone is reduced diffusely." Ex. 11 at 35. Dr. Trasmonte expressed concern with "interval worsening of prior abnormalities intracranially," adding that "[b]ecause of the exquisite symmetry of the lesions a mitochondrial process is first in the DDX [differential diagnosis] but I am keeping [the] possibility of ADEM in the list just because of the interval worsening of symptoms." *Id.* A repeat MRI produced results Dr. Trasmonte deemed "strikingly similar" to the first one performed earlier that year, and the "pattern of abnormalities" suggested to him that "an underlying metabolic/mitochondrial process" might explain K.J.'s condition, although a specific etiology remained elusive. *Id.* at 37–38. Dr. Trasmonte proposed conducting whole exome genetic testing in the search for a cause. *Id.* He also took note of concerns expressed by Ms. Whitehead that vaccination might be the reason for K.J.'s illness, although he observed that "the classic manifestation of vaccine related brain injury is ADEM," but the MRI findings were not consistent with it. *Id.* At most, K.J. had "an underlying subclinical metab[olic]/mit[oc]hondrial process and vaccination was a factor in its becoming symptomatic." *Id.*

K.J. was discharged on April 22, 2017, with a diagnosis of possible metabolic/mitochondrial disease. Ex. 11 at 38. No other records relevant to his injury reflecting treatment in 2017 have been filed in this matter.

2018 and Identification of Genetic Mutation

On January 31, 2018, K.J. underwent another brain MRI, the results of which showed "resolution of previously seen areas of acute injury to the brain," and no findings suggestive of progressive demyelination. Ex. 13 at 49. That same month, he underwent a neurological examination that was performed by Stephanie Keller, M.D. Ex. 13 at 63. Dr. Keller's exam records memorialized K.J.'s history over the prior year, and in particular his motor and feeding issues. *Id.* In reviewing K.J.'s laboratory and radiologic studies, Dr. Keller noted a "[m]utation in PHKA2 gene which is x-linked," leading her to recommend that Ms. Whitehead also be tested. *Id.* at 66. Dr. Keller's impressions following exam were (1) "spastic quadriplegic cerebral palsy" and (2) "developmental regression after [] vaccinations and illness." *Id.* at 67. K.J. was referred for a genetics consultation. *Id.*

A year later, on October 31, 2019 (and thus while this case was pending), K.J. underwent whole exome sequencing with mitochondrial DNA analysis. *See generally* Ex. 22. K.J. was identified as harboring two variants of unknown significance on the SLC19A3 gene—one inherited from each parent, and a *de novo* variant in the *TRRAP* gene. *Id.* K.J. thereafter had a genetic consultation with Dr. Juanita Neira Fresneda ("Dr. Neira"), Petitioners' testifying expert, on March 20, 2020, to review his recent genetic testing. Ex. 25-B at 1–4. Among other things, Dr. Neira assessed K.J. with most likely having "BTRBGD," which she characterized as being "a variable phenotype but consistent with developmental regression following vaccines or illness, encephalopathy, spasticity and abnormal brain MRI with hyperintensities in the caudate, putamen, thalamus and diffuse corticosubcortical areas." *Id.* at 3. Dr. Neira considered a possible BTRBGD diagnosis as being consistent with K.J.'s complete clinical picture, plus his relevant MRI and laboratory study findings. *Id.*

II. Testimony at Hearing and Expert Opinions

A. *Ms. Durenda Whitehead*

Ms. Whitehead, K.J.'s mother, was the first witness to testify at the hearing. *See generally* Tr. 7–21. She began by giving a general overview of the first thirteen months of K.J.'s life. *Id.* at 7. As she recalled, K.J. was doing very well during this time period, and that he was consistently meeting age-appropriate developmental milestones. *Id.* K.J. even began "cruising" and pulling himself up onto his feet, sometimes taking up to five steps on his own. *Id.* at 8. K.J. was also beginning to become verbal, saying a handful of words. *Id.* at 8–9. However, and while generally

a healthy baby, K.J. did have some breathing issues, such as asthma, and got sick periodically. *Id.* at 10.

Ms. Whitehead next described a well child check she had taken K.J. to on January 17, 2017 at Primary Pediatrics. Tr. at 10. At this visit, K.J. received his one-year immunizations, as well as some other immunizations he had not received earlier. *Id.* A few days later, Ms. Whitehead noticed K.J. was less active and appeared weaker. *Id.* at 11. She also noticed that he was whinier than usual and had not moved much at all that day. *Id.* at 11–12.

By Monday, January 23, 2017, K.J. would not come to Ms. Whitehead when she called out for him, and she noticed his legs trembling as he tried to stand up. Tr. at 12. After this trembling incident, Ms. Whitehead decided to take K.J. to the hospital. *Id.* K.J. was later transferred to Athens Regional hospital, where he was admitted. *Id*. He demonstrated difficulty in speaking, crawling, and eventually in the ability to hold his own head up. *Id.* at 13. At one point, K.J. was even unable to suck on anything and would gag when attempting to drink. *Id.*

Ms. Whitehead also recalled the few weeks before hospitalization, testifying that K.J. had not been sick or taken any hard falls that she felt could explain his sudden deterioration. Tr. at 14. The only thing that had been out of the ordinary had been the vaccinations he had received at his well child check on January 17, 2017. *Id.*

Ms. Whitehead also testified about K.J.'s progress over the past four years of his life. Tr. at 15. At five years old, K.J.'s function is significantly lower than his peers. *Id.* For example, K.J. is required to use a wheelchair for transportation, he has no speech abilities, and although he has redeveloped the ability to swallow on his own, he communicates using an eye gaze device because he is unable to grasp anything with his hands. *Id.* at 15–17. Ms. Whitehead expressed hope that K.J. will continue to progress, and that he eventually will be able to gain more independence. *Id.* at 17.

B. *Mr. Keynard Johnson, Sr.*

Mr. Johnson, K.J.'s father, was the second witness to testify at the hearing, and his testimony largely echoed Ms. Whitehead's testimony. *See generally* Tr. at 21–27. He recalled that K.J.'s first year of life was characterized by "walking, talking, eating, holding his own cup." *Id.* at 22. K.J. was also beginning to say words such as "mama" and "dada." *Id.*

But Mr. Johnson observed significant changes after K.J.'s vaccinations in January 2017. Tr. at 23. K.J. went from a cheerful, happy baby to slowed-down and sad looking. *Id.* At the hospital in particular, K.J. went from "a normal one-year-old to a new-born baby, really less than

a new-born baby." *Id.* at 24. K.J. was now not able to do anything, and Mr. Johnson described him as lying there "like he was almost dead." *Id.*

Over the next four years, K.J. has made progress with the help of therapy. Tr. at 24. While he still is unable to hold anything in his hands, he can now open and close them. *Id.* K.J. can also bear a little bit of his weight with the use of a walker, although he cannot stand on his own. *Id.* Mr. Johnson also testified that other than the vaccines K.J. received when he was thirteen months old, there was nothing unusual that had happened to him that might explain his sudden decline. *Id.*

C. *Petitioners' Expert—Juanita Neira Fresneda, M.D.*

Besides having treated K.J. and prepared a clinical note which was filed in this case, Dr. Neira prepared one expert report and testified at the hearing. Report, dated July 30, 2020, filed as Ex. 25 (ECF No. 28) ("Neira Rep."); Tr. 27–66; Clinical Note, filed July 30, 2020 as Ex. B (ECF No. 28).

Dr. Neira obtained a Doctor of Medicine with honors in 2008 from Fundación Universitaria de Ciencias de la Salud, Bogotá, Colombia (University of Health Sciences) Hospital San José. Curriculum Vitae, filed as Ex. A (ECF No. 28) ("Neira CV") at 1. She then completed a pediatrics residency at Woodhull Medical and Mental Health Center, Brooklyn, New York as well as a residency in Medical Genetics at Baylor College of Medicine in Houston, Texas. Neira CV at 1. Dr. Neira also completed a Medical Biochemical Genetics fellowship at Baylor College. Tr. at 28. She is licensed to practice medicine in the state of Georgia. Neira CV at 1. Currently, she is an Assistant Professor at the Department of Human Genetics at Emory University. Dr. Neira regularly sees patients in the general genetics' clinic at Emory Healthcare, focusing on treatment of pediatric patients with genetic and metabolic disorders, and also manages in-patients with genetic and inborn errors of metabolism at Children's Healthcare of Atlanta (Egleston and Scottish Rite campuses). Tr. at 28. Her publications in recent years have focused on some specific genetic syndromes impacting child development. Neira Rep. at 2.

Dr. Neira's first encounter with K.J. was as a patient in 2019, before she possessed any knowledge of this vaccine injury claim, but well after the claim had been initiated. Tr. at 29, 35. Because he was a new patient, Dr. Neira looked over K.J.'s entire medical history in order gain a better understanding of the big picture and to focus her genetic inquiries. *Id.* at 29–31. She identified no neurodevelopmental red flags or warnings within that pre-vaccination record. *Id.* at 34. Before this time, K.J. had already had extensive genetic work-ups, including a normal chromosome microarray and mitochondrial genome sequencing which only revealed a variant of uncertain significance deemed inconsistent with the patient's presentation. *Id.* at 36.

At her first visit with K.J., it was evident to Dr. Neira that he was still severely delayed for his age and exhibiting an abnormal neurologic exam. Tr. at 35. In January 2020, she ran additional genetic studies on K.J. (including whole genome sequencing and a search for metabolic derangements), which would cover every known genetic disorder in the human body. *Id*. at 36. The results showed that he possessed two variants in a gene called SLC19A3—one coming from Ms. Whitehead and one from Mr. Johnson. *Id.* at 37.

The SLC19A3 gene, she explained, codes the proteins that constitute a thiamine transporter, which is important for the mitochondria in cells responsible for energy production. *Id.* at 45. If an individual is deficient in thiamine, mitochondrial enzymes are affected because thiamine acts as a precursor for a substance called Acetyl-CoA which will start process of cellular energy production. *Id.* If this cycle is not working, the body is going to be energy depleted, causing patients to present with basal ganglia injury along with symptoms like weakness, ataxia, dysarthria, etc. *Id.* Misfunction of this thiamine transporter is thus associated with BTRBGD, or thiamine-responsive encephalopathy type-2. Neira Rep. at 2.

Dr. Neira provided some details about the nature of BTRBGD—and why it logically could be vaccine-triggered. Because BTRBGD is reasonably understood to be comparable to be an energy metabolism disorder, its phenotype or clinical presentation can be triggered by a stress or catabolic conditions in a patient, resulting in acute clinical symptoms. Tr. at 38, 43; *see* M. Alfadhel & B. Tabarki, *SLC19A3 Gene Defects Sorting the Phenotype and Acronyms: Review*, 49 Neuropediatrics 83–92 (2018), filed as Ex. D, Tab 6 on Mar. 30, 2021 (ECF No. 39) ("Alfadhel & Tabarki"). She further opined that it is well known that vaccines are stressors on the body and provoke just that kind of response. Tr. at 40; Neira Rep. at 5; Alfadhel & Tabarki at 91. She stated that since K.J. received so many vaccines at one time, and that some of the vaccines contained a live virus, it could place severe stress in his body.[3] Tr. at 40–41; Neira Rep. at 5.

K.J.'s clinical presentation and MRIs, Dr. Neira maintained, were consistent with BTRBGD. Neira Rep. at 2. His MRI findings echoed what other patients with an "SLC19A3-driven encephalopathy" have experienced (in particular the symmetric areas of abnormal signal and evidence of abnormal enhancement of the basal ganglia). *Id.* at 6. He also displayed unexplained elevated blood lactate levels at the time of his January 2017 hospitalization. *Id.*

The medical history in this case, taking into account pre- and post-vaccination events, was in Dr. Neira's view supportive of the conclusion that the vaccines K.J. received in January 2017 were responsible for the sudden manifestation of his BTRBGD. K.J. remained neurologically intact all the way until the vaccinations in January of 2017 despite several significant illnesses—plus the fact that he *already* possessed the genetic mutations required for BTRBGD. Tr. at 63–64.

[3] At least one of K.J.'s vaccines distributed on January 17, 2017—the MMR—is known to contain a live attenuated virus. *See* MMR Package Insert, *available at* https://www.fda.gov/media/75191/download.

By contrast, he had previously experienced other environmental triggers that could have provoked manifestation of his underlying genetic problem—and the fact that they did not bring on the disease in the same way that vaccination did was telling.

For example, nine months before January 2017, K.J. was seen at Children's Hospital of Atlanta in April 2016, where his presentation included a 105-degree temperature. Tr. at 63; Ex. 7 at 167–68. A few months after that, on June 9, 2016, K.J. similarly presented for treatment primarily for a breathing issue but with a report of a 101-degree fever. *Id.* at 64. Neither of these stressful events triggered sudden neurologic decline, however. *Id.* In fact, K.J. appeared to develop normally all the way until the vaccinations in January of 2017. *Id.* And immediately prior to receipt of the vaccines, he had experienced no fever or symptoms of any upper respiratory infection. *Id.* at 63–64.

Then, the week prior to presentation in the hospital, K.J. received his four-month and six-month catch-up immunizations—meaning he received seven vaccinations at one time. Tr. at 43; Neira Rep. at 3, 6. The fact that K.J. received so many at once likely caused energy depletion, which could result in the brain injuries and insults seen in K.J.'s initial MRI. Tr. at 41. Dr. Neira could not identify one particular vaccine as causal—admitting that it was out of her immediate area of expertise— but reiterated her view that the receipt of so many vaccinations in one day inevitably caused much more stress on the body than the receipt of only one. *Id.* at 39, 44; Neira Rep. at 7.

K.J.'s subsequent history showed obvious clinical deterioration. Tr. at 34; 57. In this period of time, he had an extensive workup at the hospital, with several medical teams involved, finding no additional explanation for his symptoms. Tr. at 40. Dr. Neira highlighted the fact that K.J.'s clinical symptoms came on in an acute rather than progressive manner. *Id.* at 51. Thus, he suddenly developed ataxia, and then his neurologic exam changed from baseline to having decreased muscle tone and different reflexes. *Id.* The unexpected and severe way K.J.'s symptoms presentation manifested were consistent with an immediately-prior environmental trigger—here, the vaccines.

Dr. Neira admitted that K.J.'s course was consistent with his genetic disease, regardless of the alleged vaccine trigger. Tr. at 56. Indeed, K.J.'s underlying genetic mutation was what ultimately created his symptoms – but, Dr. Neira stressed, the vaccines were responsible for setting their deleterious effects into motion. *Id.* at 57. She also explained that the "relapse" K.J. seemed to have experienced in April 2017 was not unusual, given that patients can have additional encephalopathic episodes every time there is an additional stressor. *Id.* at 59. And, in fact, K.J.'s MRI had already shown signs of necrosis, which means dead tissue of his brain. *Id.* Therefore, a subsequent more severe or more acute presentation was unsurprising, even if it was not *itself* also triggered by vaccination in the same way as K.J.'s initial presentation. *Id.* But once the vaccines

had first "unmasked" K.J.'s underlying BTRBGD, further deterioration was to be reasonably expected. *Id.*

D. *Petitioners' Expert—James E. Carroll, M.D.*

Dr. Carroll provided two expert reports in this matter but did not testify. *See* Report, filed Sept. 25, 2019 as Ex. 20 (ECF No. 20-3) ("First Carroll Rep."); Report, filed April 3, 2020 as Ex. 21 (ECF No. 22) ("Second Carroll Rep.").

Dr. Carroll obtained his bachelor's degree and his medical degree from the University of Louisville. First Carroll Rep. at 1. He completed a pediatrics internship and residency at Louisville Children's Hospital. *Id.* Dr. Carroll served as a Lt. Commander in the U.S. Navy at Bethesda Naval Hospital after which he completed a pediatric neurology fellowship at the University of Colorado Medical Center. *Id.* He also completed a neuromuscular diseases fellowship at Washington University, St. Louis, Missouri. *Id.* Dr. Carroll is board certified in neurology with special competence in child neurology and was an associate professor of Pediatrics and Neurology at Washington University and at the Medical College of Georgia. *Id.* at 2. Since 1999, Dr. Carroll has been a research biologist at the Veterans Administration Hospital in Augusta, Georgia. *Id.* He has authored and co-authored approximately one hundred peer-reviewed medical journal articles and several book chapters and monographs. *Id.* He has maintained an active pediatric neurology clinic, seeing patients both in office and inpatient. *Id.*

Dr. Carroll's first report was prepared and filed before the discovery of K.J.'s genetic mutation. Based on a review of the relevant medical records, Dr. Carroll opined that the MMR vaccine caused KJ to experience an encephalitis, manifesting in the neurologic symptoms documented in the record. First Carroll Rep. at 10–12. He expressly disclaimed that any genetic defect could explain this proposed encephalitis. *Id.* at 12. Although Petitioners have not formally abandoned the opinion expressed therein, I find that the discovery of the mutation, coupled with both testifying expert's embrace of the BTRBGD diagnosis, greatly reduces the evidentiary value of this report, and thus do not give it further discussion or attention.

In his second, shorter report, Dr. Carroll opined that it was unlikely that K.J. suffered from a progressive genetic syndrome, such as Leigh syndrome,[4] but that whatever he had experienced was likely triggered by the January 2017 vaccines. Second Carroll Rep. at 1. He also took aim at

[4] Leigh syndrome is a subacute necrotizing encephalomyelopathy. *Dorland's Illustrated Medical Dictionary* (33d ed. 2020) at 531 [hereinafter *Dorland's*]. Respondent's Expert, Dr. Raymond, explained Leigh syndrome's clinical features, which include "ataxia, hypotonia, spasticity, optic atrophy, nystagmus, ophthalmoplegia (eye movement abnormalities), abnormal respiration patterns, and the loss of previously-acquired skills. There is often lactic acidosis of the blood, cerebrospinal fluid, and urine, although this is not always present. The classic form of Leigh has onset in infancy and is rapidly progressive to death in 5 years after onset." *See* Report, filed Jan. 1, 2020 as Ex. A (ECF No. 21-1) ("First Raymond Rep.") at 8–9.

contentions in the reports of Respondent's expert about K.J.'s course. *Id.* at 2. And he briefly referenced the genetic testing results, accepting that they corroborated the conclusion that K.J.'s symptoms were attributable to a "thiamine deficiency" associated with the SLC19A3 genetic variant, but maintained nevertheless that vaccination could trigger it, referencing treater evidence in support. *Id.* at 4–5.

E. *Respondent's Expert—Gerald Vincent Raymond, M.D.*

Dr. Raymond provided two expert reports in this matter and testified at the hearing. *See* Report, filed Jan. 1, 2020 as Ex. A (ECF No. 21-1) ("First Raymond Rep."); Report, filed Sept. 14, 2020 as Ex. C (ECF No. 30-1) ("Second Raymond Rep."); Tr. 66–167. Dr. Raymond opined that K.J.'s progressive genetic neuro-degenerative disorder (reasonably diagnosed as BTRBGD) was not caused or exacerbated by his January 17, 2017 vaccinations, but rather constituted a condition he was born with. Tr. at 126.

Dr. Raymond obtained his medical degree from the University of Connecticut. Curriculum Vitae, filed as Ex. B (ECF No.21-6) ("Raymond CV"); Tr. at 66. He then did a residency in pediatrics at Johns Hopkins followed by a three-year residency in neurology with special qualifications in child neurology at Massachusetts General Hospital. *Id.* at 66–67. Dr. Raymond also completed a fellowship at the Université Catholique do Louvain in Brussels specializing in developmental neuropathology. *Id.* at 67. Following his fellowship, he returned to Massachusetts General and Harvard where he completed a three-year fellowship in genetics and teratology. *Id.* Dr. Raymond is board certified in neurology with special qualifications in child neurology as well as clinical genetics. Raymond CV at 1. Currently, Dr. Raymond is employed at Johns Hopkins University Hospital where he is a professor of genetic medicine and neurology. Tr. at 67. Approximately 75 to 80 percent of his work is clinical, attending to inpatient service and working in the Kennedy Krieger neurogenetics clinic. *Id.* at 67–68. Dr. Raymond has also published numerous peer-reviewed articles in pediatric neurology and clinical genetics. *Id.* at 68.

Dr. Raymond began his testimony by giving some basic background on the relationship between the SLC19A3 gene and thiamine uptake in the body (and the brain in particular). Tr. at 66. As a general matter, a gene is an inherited unit composed of DNA, with a sequence of nucleotides that encodes a protein or, in some cases, nonprotein RNA. *Id.* Thiamine, a cofactor sometimes referred to as a vitamin, is needed for the enzymatic action of a variety of biochemical occurrences in the body (and thus its absence in the body has biochemical significance). *Id.* at 73. Thiamine is particularly important in glucose metabolism, mitochondrial energy production (via the Krebs cycle), and several other key energy-releasing processes. *Id.* Thus, without thiamine the body will have a defect in the alpha-oxidation of certain forms of lipids interfering with the body's ability to utilize energy from lipid stores. *Id.*

The SLC19A3 gene, Dr. Raymond explained, is essential for assisting the transport of thiamine across cell membranes and into the brain. Tr at 72–73. As a result, a defect or mutation in that gene impairs thiamine transport, and in so doing results in harm. Such a thiamine-related genetic disorder can have a variety of presentations. *Id.* at 77. It can, for example, have an infantile presentation, which will appear like Leigh syndrome, or it can have an adult form which can look a little bit like Wernicke's encephalopathy.[5] *Id.* The classic presentation, however, is referred to as BTRBGD, and is generally deemed a progressive neurological disease responsive to biotin (vitamin B_7) and thiamine. *Id.* at 78.[6] Its presumed pathogenesis is attributable to a defective thiamine transporter, likely the result of a genetic mutation. Tr. at 86. Since such transporters have particular significance to ensuring energy production in the brain, BTRBGD will typically manifest with symmetric basal ganglia disease, while also involving other areas of the brain. *Id.* at 86–87; Second Raymond Rep. at 7.

Dr. Raymond offered and discussed two items of literature that shed light on BTRBGD's presentation or possible causes. *See, e.g.*, P. Ozand et al., *Biotin-Responsive Basal Ganglia Disease: A Novel Entity*, 121 Brain 1267–79 (1998), filed as Ex. C, Tab 6 on Sept. 14, 2020 (ECF No. 30-7) ("Ozand"). Ozand considered ten individuals, ranging from ages two through seven years old, and their disease course, and concluded that BTRBGD commonly appears as a subacute encephalopathy but progresses into dystonia and quadriparesis.[7] Ozand at 1267. These symptoms were found to disappear within a few days, however, if biotin was administered, with neurological sequelae reappearing within one month if biotin was discontinued. *Id.* Ozand's authors could not identify the disease's etiology but stressed the importance of biotin therapy in reversing and preventing symptom progression. *Id.* Dr. Raymond made a point of highlighting one asymptomatic patient in the study who was identified because of a symptomatic sibling. Tr. at 78. An MRI for this patient revealed evidence of basal ganglia disease despite the lack of symptoms – and Dr. Raymond deemed this significant because it suggested to him that BTRBGD would likely progress subacutely, and prior to a presumed trigger. *Id.*

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[5] Wernicke encephalopathy may often intertwine with Wernicke-Korsakoff syndrome, a "behavioral disorder caused by thiamine deficiency, most commonly due to chronic alcohol abuse and associated with other nutritional polyneuropathies. Wernicke encephalopathy (confusion, ataxia of gait, nystagmus, and ophthalmoplegia) occurs as an acute attack and is reversible, except for some residual ataxia or nystagmus, by administration of thiamine; Korsakoff's syndrome (severe anterograde and retrograde amnesia) may occur in conjunction with Wernicke encephalopathy or may become apparent later; only about 20 per cent of patients recover completely from the amnesia." *Dorland's* at 1824.

[6] Dr. Raymond also discussed how treatment of the disorder has evolved over time. Tr. at 81. While initially researchers believed that biotin was the only, or at least preferred, treatment, more recent research had established that thiamin alone is sufficient. *Id.* at 81–82.

[7] Dystonia is "dyskinetic movements due to disordered tonicity of muscle." *Dorland's* at 576. Quadriparesis, also called tetraparesis, is "muscular weakness affecting all four limbs" *Id.* at 1877.

By contrast, another study cited by Dr. Raymond observed a preceding, potentially-triggering event, including viral infection or vaccination, in six patients shortly before onset of early-infantile encephalopathies due to SLC19A3 gene mutation. Tr. at 78; S. Kevelam et al., *Exome Sequencing Reveals Mutated SLC19A3 in Patients with an Early Infantile, Lethal Encephalopathy*, 136 Brain 1534–1543 (2013), filed as Ex. C, Tab 3 on Sept. 14, 2020 (ECF No. 30-4) ("Kevelam"). But despite the suggestion in Kevelam that many of these encephalopathies were triggered by vaccinations, Dr. Raymond proposed that its authors may more likely have relied on a temporal association rather than proof of a causal relationship. Tr. at 91;[8] Kevelam at 1542.

To support his contention that triggers were not critical in the development of SLC19A3-related disorders like BTRBGD, Dr. Raymond referenced some animal studies. Tr. at 87–89; K. Suzuki et al., *High-Dose Thiamine Prevents Brain Lesions and Prolongs Survival of SLc19a3-Deficient Mice*, PLoS ONE 12 (6): e0180279, http://doi.org/10.1371/journal.pone.0180279 1-17 (June 30, 2017), filed as Ex. C, Tab 19 on Sept. 14, 2020 (ECF No. 31-9) ("Suzuki"); K. Vernau, et al., *Genome-Wide Association Analysis Identifies a Mutation in the Thiamine Transporter 2 (SLC19A3) Gene Associated with Alaskan Husky Encephalopathy*, PLOS ONE 8(3): e57195. Doic.10.1371/journal.pone.0057195 (March 4, 2013), filed as Ex. C, Tab 18 on Sept. 14, 2020 (ECF No. 31-8) ("Vernau"). Vernau demonstrated that a certain form of encephalopathy was common to Alaskan huskies, who experienced deficiencies in energy production due to thiamine transporter genetic deficiencies comparable to what humans also experienced. Second Raymond Rep. at 7; Vernau at 5. Dr. Raymond opined, however, that Vernau and Suzuki established not only that thiamine was effective in treating the condition, but also that the BTRBGD-like conditions came about *regardless* of trigger (including vaccination). Tr. at 88, 89.

Overall, Dr. Raymond did not deny that literature referenced by Petitioners suggested that a variety of environmental stressors—including vaccines—could trigger "acute episodes" for children with BTRBGD. Second Raymond Rep. at 7–8; Kevelam at 1538; J.D. Ortigoza-Escobar et al., *Thiamine Deficiency in Childhood with Attention to Genetic Causes: Survival and Outcome Predictors*, 82 Annals Neurology317, 320 (Sept. 10, 2017), filed as Ex. 25, Tab E on July 30, 2020 (ECF No. 28) ("Ortigoza-Escobar"). Indeed, he acknowledged that *his own literature* emphasized that "under physiologic stresses" thiamine transport deficiencies attributable to underlying SLC19A3 genetic mutations could manifest. Second Raymond Rep. at 7 ("decline and clinical manifestations may occur when the energy required exceeds the background levels and there is an inability of the altered forms of the transporter to undergo the normal stress-induced expression and functional compensation"); A. Schanzer et al., *Stress-Induced Upregulation of SLC19A3 is Impaired in Biotin-Thiamine-Responsive Basal Ganglia Disease*, 24 Brain Pathol. 270–79 (2014), filed as Ex. C, Tab 20 (ECF No. 31-10) ("Schanzer"). Schanzer does not discuss vaccination in

[8] Dr. Raymond also discounted the apparent conclusions of Kevelam's authors because of the retrospective aspect of the study, plus the fact that no data was provided that would shed light on either (a) the specific vaccines at issue, or (b) the timeframe from vaccination to onset. Tr. at 91.

particular, but does opine that a febrile illness or infection could constitute sufficient stress to lead to acute manifestations. Schanzer at 270. Dr. Raymond nevertheless maintained that not enough was known to conclude that vaccination could in fact act as such an environmental impetus. Second Raymond Rep. at 8, 10.

Dr. Raymond went on to consider K.J.'s medical history and the diagnoses contained therein. In the course of the litigation, his opinion changed a bit as to the proper diagnosis, after the results of K.J.'s genetic testing were obtained in the fall of 2019. His first report was prepared in response only to Dr. Carroll's initial report, and it largely proposed that K.J. suffered from an unidentified "progressive genetic neurodegenerative disorder" that Dr. Raymond felt was comparable to Leigh syndrome. First Raymond Rep. at 13. By the time of his second report, however, Dr. Raymond had the benefit of Dr. Neira's report plus the genetic testing results. Now, he deemed K.J.'s course and presentation "consistent with" the disorder associated with the SLC19A3 variant (in effect, BTRBGD). Second Raymond Rep. at 6, 8.

Dr. Raymond next considered the pre-vaccine evidence of K.J.'s neurologic dysfunction. For example, K.J. failed his newborn hearing screening. Tr. at 142–43; Ex. 5 at 33. Additionally, K.J. had been hospitalized in June 2016 for treatment of acute bronchiolitis and laryngomalacia. Tr. at 96. K.J. also had an abnormal swallow study at the time, and Dr. Raymond deemed it significant because it could indicate preexisting neurologic dysfunction—possibly associated with low muscle tone and hypotonia. *Id.* at 96–97. He admitted on cross-examination, however, that there was no pre-vaccination evidence of clear BTRBGD onset, despite the demonstration of potential environmental triggers, like fever or other illnesses, having occurred. *Id.* at 159–61.

Dr. Raymond disagreed with certain post-vaccination evidence that seemed to suggest treater support for Petitioners' claim. He specifically took issue with a radiologist's impression of a brain and complete spine MRI performed on January 24, 2017. Tr. at 103. While he agreed with the description of what the imaging literally depicted, he disputed the technician's impression that K.J. possibly suffered from "postvaccination autoimmune encephalitis[.]" *Id.* at 104; Ex. 7 at 3139–3140. Dr. Raymond ultimately proposed that the progressive nature of K.J.'s condition precluded the conclusion that it could be brought on acutely. Second Raymond Rep. at 8–9.

In addition, although Dr. Raymond accepted the existence of case reports identifying instances of post-vaccination ADEM as a comparable injury, he denied that "postvaccination autoimmune encephalitis" was a legitimate pathologic entity. Tr. at 105. Dr. Raymond also stressed that there was no evidence in the medical records that showed K.J. was at this time suffering any signs of encephalopathy—either acute or subacute. *Id.* at 105–107; 116. And while there are various notes by K.J.'s treaters from early in his treatment including in their differentials a possible link to vaccination (i.e. "metabolic process like a mitochondrial disease possibly activated by vaccines" (Ex. 7 at 3276; Tr. at 111), treaters were generally moving away from the

consideration—which is evidenced by the pursuit of a variety of other possible etiologies. Tr. at 112.

Finally, Dr. Raymond highlighted K.J.'s period of improvement once he was discharged from the hospital in February 2017 (after responding well to thiamine treatment), contrasting it to his subsequent April 2017 regression, featuring significant loss of abilities plus significantly worsened presentation on MRI. Tr. at 122. Dr. Raymond specifically proposed that this occurrence was the product of a cessation of the thiamine treatments that had initially been so beneficial for K.J. Second Raymond Rep. at 8–9. This assertion was not, however, supported by any record cite establishing thiamine cessation to have in fact occurred, and it appears rather than Dr. Raymond assumed this to be the case based on statements made by Petitioners to a speech language pathologist in March 2017 about their feeding practices for K.J. at that time.

By this point, Dr. Raymond opined, K.J. was "manifesting some degree of encephalopathy," since he was less responsive, and his decline was confirmed by an abnormal EEG result. *Id.* at 122. K.J. had now experienced sever developmental loss and was described on exam as having spastic quadriplegia. *Id.* at 124. This relapse was significant to Dr. Raymond, because he felt it demonstrated that K.J.'s decline was overall progressive rather than acute (as might be expected if the vaccines has truly triggered the BTRBGD). *Id.* at 125; First Raymond Rep. at 8.

III. Procedural History

After the case's initiation in October 2018, Petitioners filed medical records supporting the claim along with the Statement of Completion. ECF No. 8. Respondent's Rule 4(c) Report was filed in April 2019, and then shortly thereafter the case was reassigned to me. ECF No. 12. In January 2020, Respondent filed Dr. Raymond's expert report. ECF No. 21. Later that year, in April, Petitioners filed Dr. Carroll's expert report. ECF No. 22. I set the matter for a hearing, to be held March 4–5, 2021. ECF No. 26. Prior to the hearing, Petitioners filed Dr. Neira's expert report in July 2020, and Respondent filed Dr. Raymond supplemental expert report in September 2020. ECF Nos. 28, 30. The trial occurred as scheduled, and both parties filed post hearing briefs in July 2021. ECF Nos. 40–41. The matter is now ripe for resolution.

IV. Applicable Law

A. *Standards for Vaccine Claims*

To receive compensation in the Vaccine Program, a petitioner must prove that: (1) they suffered an injury falling within the Vaccine Injury Table (i.e., a "Table Injury"); or (2) they suffered an injury actually caused by a vaccine (i.e., a "Non-Table Injury). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also*

Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano*, 440 F.3d at 1320. To bring a successful Table claim, the petitioner must make a precise factual showing sufficient to meet the Table's relevant definitions, as set forth in the Table's "Qualifications and aids to interpretation" ("QAIs"). Section 14(b). If successful, the petitioner need not establish vaccine causation, as it is presumed if the Table requirements for a particular claim are met. Section 14(a). In this case, the Petitioners assert both a Table and non-Table claim.

For both Table and Non–Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (explaining that mere conjecture or speculation is insufficient under a preponderance standard). On one hand, proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). But on the other hand, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non–Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health and Hum. Servs.,* 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury." Each *Althen* prong requires a different showing and is discussed in turn along with the parties' arguments and my findings.

Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

However, the Federal Circuit has *repeatedly* stated that the first prong requires a preponderant evidentiary showing. *See Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1360 (Fed. Cir. 2019) ("[w]e have consistently rejected theories that the vaccine only "likely caused" the injury and reiterated that a "plausible" or "possible" causal theory does not satisfy the standard"); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir.

2010); *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1350 (Fed. Cir. 2010). This is consistent with the petitioner's ultimate burden to establish his overall entitlement to damages by preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted). If a claimant must *overall* meet the preponderance standard, it is logical that they be required also to meet each individual prong with the same degree of evidentiary showing (even if the *type* of evidence offered for each is different).

Petitioners may offer a variety of individual items of evidence in support of the first *Althen* prong, and are not obligated to resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). No one "type" of evidence is required. Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Andreu,* 569 F.3d at 1380. But even though "scientific certainty" is not required to prevail, the individual items of proof offered for the "can cause" prong must *each* reflect or arise from "reputable" or "sound and reliable" medical science. *Boatmon,* 941 F.3d at 1359–60.

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742,

749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Hum. Servs.*, No. 06–522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356–57 (2011), *aff'd without opinion*, 475 F. App'x. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 2013 WL 1896173 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11–355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Law Governing Analysis of Fact Evidence*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs.*, 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical

professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health*

& Hum. Servs., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too

great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. *Consideration of Medical Literature*

Both parties filed medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

Both testifying experts agreed that K.J. possesses a genetic variant that has played a role in causing him to experience BTRBGD—a thiamine-transporting deficiency resulting in clear neurologic deficits and other negative symptoms. They disagreed, however, whether vaccines can cause BTRBGD to manifest, and also whether the facts of this case are sufficient to meet the evidentiary requirements relevant to both Table and non-Table Program claims. Below, I address the disputed issues of fact and law.

I. *Petitioners Have Not Established a Table Encephalopathy*

Previously in the course of litigation I informed Petitioners that I did not deem their Table MMR vaccine-encephalopathy claim to be particularly strong. Petitioners, however, opted to

persist in asserting it as a basis for liability. Now, having heard the evidence at trial, I find that my preliminary views were correct, and the Table claim warrants dismissal.

According to the QAI applied to vaccine Table claims, a vaccinee is considered to have suffered a Table encephalopathy if he or she manifests an injury encompassed in the definition of an "acute" encephalopathy within the appropriate time period, *and then* a "chronic" encephalopathy is present for more than six months after the immunization. 42 C.F.R. § 100.3(b)(2) (emphasis added). In accordance with the QAI, an acute encephalopathy without seizure for infants under 18 months old (which would here include K.J.) must be sufficiently severe to require hospitalization (regardless of whether the vaccinee is actually hospitalized), and it must manifest within 5 to 15 days of vaccination. 42 C.F.R. §§ 100.3(a) and (b)(2)(i)(1). A chronic encephalopathy exists if the change in mental state that began with the acute encephalopathy persists for at least six months. 42 C.F.R. § 100.3(d)(1)(i). However, there is the express caveat that

> Individuals who return to their baseline neurologic state, as confirmed by clinical findings, within less than 6 months from the first symptom or manifestation of onset or of significant aggravation of an acute encephalopathy or encephalitis shall not be presumed to have suffered residual neurologic damage from that event; any subsequent chronic encephalopathy shall not be presumed to be a sequela of the acute encephalopathy or encephalitis.

42 C.F.R. § 100.3(d)(1)(ii).

Petitioners cannot meet these strict requirements for their Table claim. First, and most importantly, the record clearly establishes that K.J.'s post-vaccination symptoms manifested *outside* the 5-15 day timeframe. The record establishes onset of K.J.'s weakness, lethargy, and difficulty moving on January 21, 2017 – four days post-vaccination. Ex. 7 at 3107. This is too soon to meet the Table requirement, even if by a single day. *See generally Greene v. Sec'y of Health & Hum. Servs.*, No. 11-631V, 2019 WL 4072110, at *18 (Fed. Cl. Spec. Mstr. Aug. 2, 2019) (noting that a risk interval concept to expand the window of a Table Claim for a brachial neuritis injury after receipt of a tetanus vaccine beyond the six-week timeframe by one day did not support a medically acceptable onset timeframe), *mot. for review den'd,* 146 Fed. Cl. 655 (2020), *aff'd,* 841 F. App'x 195 (Fed. Cir. 2020).

Second, there is an absence of treater support for the conclusion that K.J.'s presentation in January 2017 reflected the existence of an acute encephalopathy or encephalitis (either of which are subject to the same Table requirements. 42 C.F.R. § 100.3(a)(II)(B)). While the possibility of encephalitis was included in his initial differentials, treaters ultimately opined he more likely suffered from some form of mitochondrial/energy production disorder – and in fact his BTRBGD

is far more consistent with such a preliminary diagnosis than an acute encephalopathy. Ex. 7 at 3197. Finally, even if K.J.'s initial symptoms were found to be the product of an acute encephalopathy, the record does not allow me to conclude his subsequent state resulted from an ongoing chronic encephalopathy. Indeed, he came back to baseline in terms of symptoms not long after his first manifestations of symptoms. Ex. 7 at 56. Rather, he had a neurologic disorder attributable to an underlying genetic variant that caused concerning and damaging symptoms – and that harm is not "encephalopathic," as far as the Table goes, simply because it involves the brain.

Accordingly, Petitioners' Table claim is dismissed.

II. *Petitioners Have Established Causation in Fact*

Petitioners' inability to meet the requirements of their Table claim leaves only a causation-in-fact claim that the multiple vaccines K.J. received in January 2017 somehow precipitated the manifestation of a neurologic disorder otherwise attributable to the SLC19A3 genetic variant. Although Petitioners' showing was far from robust, the evidence was sufficiently close for me to find that the preponderant test for establishing entitlement was met overall.

A. *Althen* Prong One

The evidence offered for the contention that vaccines could trigger a child's preexisting SLC19A3 mutation by causing stress was quite thin – but just preponderant enough to meet Petitioners' burden.[9] Dr. Neira, a qualified geneticist as well as one of K.J.'s treaters, opined about the causal role of the vaccines – and her opinion was backed up by several items of literature, as well. Alfadhel & Tabarki at 83; Kevelam at 1542; Ortigoza-Escobar at 321; Schanzer at 270. In effect, Petitioners seem to allege that the vaccines K.J. received would (likely via some innate response, rather than a longer-term adaptive response, in which a vaccine's specific antigens would immunologically cause a cross-reaction) set the thiamine transport deficiency into motion.

Admittedly, this is *not* a case where the evidentiary support for the claimant's causation theory easily crosses the preponderance "line." The statements from articles like Ortigoza-Escobar or Kevelam associating vaccination to BTRBGD are facially conclusory, with little background information provided to evaluate their basis or ascertain if the risk is higher or lower for different vaccines. But at the same time, this is also *not* a case where Respondent can do more than declaim

[9] In so concluding, I do not adopt Petitioners' erroneous contention that mere plausibility is the standard to be applied when evaluating success as to the first *Althen* prong. The Federal Circuit has consistently reiterated that the first prong must meet the preponderant standard. *Boatmon*, 941 F.3d at 1360; *McCollum v. Sec'y of Health & Hum. Servs.*, 760 F. App'x 1003, 1007 (Fed. Cir. 2019).

that Petitioners have not *themselves* proven their case – and while such a defense can have substance when the claimant over-relies on generalities, it does not invariably succeed.

More significantly, the limited scientific knowledge base pertaining to BTRBGD generally is also harmful to Respondent's defense. The experts herein agreed that BTRBGD has its origins as a genetic mutation causing thiamine transport malfunction, and resulting in turn in energy production deficits. But can it be concluded that when the relevant mutation exists, the result is preordained – or can manifestation of the error caused by mutation be hastened, worsened, or simply "unmasked" after an environmental stimulation, like a vaccine? In somewhat comparable Program cases (such as seizure disorders, like Dravet syndrome, attributable to the SCN1A genetic mutation), there exists direct, on-point literature suggesting that the anticipated disease course otherwise attributable to the mutation/variant is *not* impacted by vaccination—even if vaccination can temporarily produce a symptoms "flare." *See, e.g.*, *Oliver v. Sec'y of Health & Hum. Servs.*, No. 10-394V, 2017 WL 747846 (Fed. Cl. Spec. Mstr. Feb. 1, 2017), *mot. for review den'd,* 133 Fed. Cl. 341 (2017), *aff'd*, 900 F.3d 1357 (Fed. Cir. 2018). Indeed, the special master in *Oliver* found that the causal link between the relevant genetic mutation and the seizure disorder was directly supported by literature that *discounted* the importance of an intervening environmental factor like vaccination. *Oliver,* 2017 WL 747846, at *15. Such rebutting evidence was thus strong enough to overcome the claimant's position that a vaccine played some role in sparking symptoms unquestionably linked to the mutation. *See Deribeaux ex rel. Deribeaux v. Sec'y of Health & Hum. Servs.*, No. 05-306V, 2011 WL 6935504, at *32 (Fed. Cl. Spec. Mstr. Dec. 9, 2011)*, review denied, decision aff'd*, 105 Fed. Cl. 583 (2012), *aff'd,* 717 F.3d 1363 (Fed. Cir. 2013).

Here, no such rebuttal evidence has been offered (and may well not even yet exist for the SLC19A3 mutation) other than Dr. Raymond's testimony. And his opinion seemed to over-rely on his determination that there was an absence of scientific certainty favoring vaccine causation – an evidentiary standard inapplicable to Program claims, even if a medical professional or scientist would remain personally unconvinced (and reasonably so) by a special master's entitlement decision favoring a particular theory. Indeed, Dr. Raymond freely acknowledged that a *variety* of environmental stressors were understood by medical science to be capable of taxing an affected individual's thiamine transport system sufficiently to cause manifestation of BTRBGD, and cited reliable literature for this point. *See* Schanzer at 278–79. And he noted in his second report that vaccination *had been implicated* in triggering clinical manifestations of BTRBGD. Second Raymond Rep. at 7. He thus could not credibly deny that the causation theory offered in this case exceeded mere plausibility.

Time and again, special masters are reminded that close calls are to be decided in a petitioner's favor. *Roberts. v. Sec'y of Health & Hum. Servs*., No. 09-427V, 2013 WL 5314698, at *10 (Fed. Cl. Spec. Mstr. Aug. 29, 2013). That is the case not only when both sides offer numerous items of persuasive and compelling evidence, but also where the evidence is quite limited (often

because the disease process at issue is novel and/or under-studied). *Andreu*, 569 F.3d at 1378–79. Although future cases may present updated evidence on the topic that weighs in favor of a different outcome, the evidence presented *in this case* is sufficient to find that the "can cause" prong has been met.

B. *Althen* Prong Two

I also find just enough support to establish a logical sequence of cause and effect from vaccine to injury. Both the record itself and Dr. Neira's testimony bulwark this conclusion. The Program recognizes that treating physicians are in a good position to opine that a vaccination was the reason for the injury. *Capizzano,* 440 F.3d at 1326. Dr. Neira provided treatment to K.J., demonstrated awareness of his clinical progression in her report, and engaged in an inquiring effort to identify the cause and nature of K.J.'s symptoms by running whole genome sequencing tests to cover every known genetic disorder in the human body. Thus, Dr. Neira's view that the stress induced by the vaccines caused a manifestation of BTRBGD is entitled to additional weight in favor of Petitioners' claim. Moreover, Dr. Trasmonte also allowed for this possibility as well (so Dr. Neira was not the sole treater to propose an association between vaccine and injury). Ex. 11 at 37–38.

Respondent did attempt to bulwark his position that the genetic mutation best explained K.J.'s BTRBGD by highlighting the fact that he experienced a relapse of sorts in April 2017. Ex. 11 at 33. Dr. Raymond speculates that this occurred because K.J. was no longer receiving thiamine, as he did earlier that year, and this shows the kind of *actual* environmental impacts that would trigger clinical manifestations. But this argument has two deficiencies.

First, the experts agreed that a thiamine transport deficiency (attributable to the genetic mutation directly) precipitates symptoms. But this does not mean that *other* environmental factors could not *first* stress the system sufficiently to cause the condition's general unmasking (by instigating the transport issue), and thus initiating the overall problem.[10] Indeed, as Petitioners noted, K.J. experienced other potential stressors prior to vaccination, but those environmental impacts did not similarly cause the same clinical symptoms. Ex. 7 at 167; Neira Rep. at 6; Second Raymond Rep. at 10. And as noted above, Dr. Raymond himself offered literature that suggested (albeit in a somewhat conclusory fashion) vaccines could have this impact on someone with BTRBGD.

Second, Dr. Raymond's contention regarding the cause of the relapse is not fully corroborated by the record. Although there is evidence that certain feeding-related matters, like a

[10] However, to the extent incomplete or inadequate treatment might best explain some of K.J.'s post-vaccination symptoms flares or relapses, such matters do bear on recoverable damages, and I will take such issues into account in the damages phase of the case.

feeding tube, had been suspended by Petitioners prior to this relapse, the record does not allow me to conclude that thiamine-specific treatments had been suspended – or if so when. His speculation on this point was certainly reasonable (as was his suspicion that K.J.'s pre-vaccination health issues could be harbingers of his later BTRBGD) – but it was not substantiated by record evidence, and thus is not enough to go on for purposes of deciding this case.

C. *Althen* Prong Three

Neither side has presented a particularly powerful case regarding the "medical acceptability" of the timeframe between K.J.'s vaccinations and onset of his BTRBGD initial symptoms. Petitioners' experts seemed to emphasize the close temporal relationship between K.J.'s vaccines and the onset of his symptoms (First Carroll Rep. at 11; Neira Rep. at 2–3)—something understood in the Program to be an insufficient basis for finding the third *Althen* prong satisfied. *de Bazan,* 539 F.3d at 1352. But in attacking this aspect of Petitioners' showing, Dr. Raymond predominantly relied on record evidence of K.J.'s intervening improvements that occurred during March and April of 2017. First Raymond Rep. at 12–13. This contention largely ignores the primary question posed by this part of the entitlement test: whether the relationship between *initial* symptoms and vaccination was acceptable in light of the alleged causation theory. *de Bazan,* 539 F.3d at 1352.

A careful evaluation of Petitioners' expert opinions, however, allows me to conclude that this prong (like the two before it) was met, if barely. Dr. Neira was both a qualified geneticist and treater, and I give weight to her opinion (based on both the genetic testing and K.J.'s overall medical history) that K.J.'s BTRBGD was likely sparked in the four days after vaccination. Neira Rep, at 6; *see also* Prehearing Brief at 8–9. Her opinion gained additional heft from the medical literature offered in this case. Alfadhel & Tabarki at 91; Kevelam at 1539; Ortigoza-Escobar at 320. Although such literature does not contain a specific temporal prediction of expected onset of BTRBGD after an environmental stressor, it is consistent with the conclusion that onset could reasonably occur within days. Kevelam at 1541–42 (indicating that when there is a cerebral MRI abnormality, such as in BTRBGD cases, it indicates *rapid* onset) (emphasis added). And the theory offered in this case is consistent with other vaccine cases in which the stress impact of a large number of vaccines administered at once, or a particularly potent vaccine, might (via the body's innate immune reaction) cause a reaction within days. *See, e.g.*, *Halverson v. Sec'y of Health & Hum. Servs.,* No. 15-227V, 2020 WL 992588, at *27 (Fed. Cl. Spec. Mstr. Feb. 4, 2020) (finding that there was a temporal relationship between receipt of the fluzone vaccine and the developing injury that caused petitioner's death four days after vaccination); *see also Gerhardt v. Sec'y of Health & Hum. Servs.*, No. 9-180V, 2014 WL 4712690, at *12 (Fed. Cl. Spec. Mstr. Aug. 29, 2014) (petitioner established a proximate temporal relationship between receiving five vaccines and the onset of his neurologic symptoms three days later).

Respondent in reaction did not rebut this conclusion. Thus, I find the third *Althen* prong was met.

CONCLUSION

As I have noted in other cases, Vaccine Program claimants can prevail *despite* the fact that considerable uncertainty remains that they are in fact correct. "More likely than not" is an inexact measurement of causation, to say the least.

Because of the rarity of the underlying genetic condition and lack of substantial research into its pathophysiology, there remains much to learn about BTRBGD. It is conceivable that a stronger showing by Respondent, relying on more up-to-date research into how this disease progresses and what causes it to manifest in the first place, could cast doubt on my conclusion herein – and result in an entirely different outcome in future cases. However, I find sufficient preponderant evidence was offered *in this case* to support Petitioners' contention that the receipt of several vaccines at one time could trigger SLC19A3 variants, and did so here. Thus, I must conclude on the basis of Petitioners' showing that they have met their evidentiary burden, even if that showing was not overwhelming.

In order to guide the parties through the damages phase of the action, a separate damages order will issue.

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master